EXHIBIT A

SUPPLEMENTAL/REBUTTAL
EXPERT REPORT OF VICTOR L. ROGGLI, M.D.

Report for David T. Austern,
Future Claimants' Representative

In re W.R. Grace & Co., *et al.*

July 31, 2007

In this supplemental/rebuttal expert report, I comment on the medical criteria upon which Dr. B. Thomas Florence relies in making his estimate of the value of W.R. Grace & Co.'s (collectively, "Grace") pending and future asbestos personal injury claims.

Dr. Florence assumes a number of medical criteria upon which the value of pending and future asbestos personal injury claims against Grace is based. In my opinion, as discussed below, these criteria are not medically sound, and any estimate based on these criteria would be flawed. I address certain of these criteria in the body of this report, and in the course of doing so, I respond to the opinions of other Grace experts on whom Dr. Florence appears to rely for the medical criteria he is using as a screen. Those experts are Elizabeth Anderson, PhD, Gordon Bragg, PhD, Steven E. Haber, MD, FCCP, Dr. Daniel A. Henry, Grover M. Hutchins, MD, Richard J. Lee, PhD, Peter S J Lees, PhD, CIS, Drs. M. Laurentius Marais and William E. Wecker, Suresh Moolgavkar, MD, PhD, Joseph V. Rodricks, PhD, and David Weill, MD.

Minimum Exposure Criteria

The minimum exposure criteria set forth at point 2 of page 2 of Dr. Florence's Report are invalid. Under Grace's minimum exposure criteria, only a worker who personally mixed or installed Grace asbestos-containing products is eligible for compensation. However, as outlined in my prior report dated June 11, 2007, a substantial fraction of mesothelioma cases occur in bystanders, and these patients have considerably elevated lung burdens of asbestos.

Furthermore, the minimum criteria exclude workers who may have *removed* Grace products, another important potential source of exposure. The minimum exposure criteria also exclude claimants who become sick based on household contacts of asbestos workers. Such contacts are a major source of asbestos-related disease among women as detailed in my prior report.

The minimum exposure criteria that Dr. Florence assumes appear to be based on threshold benchmarks established by other Grace experts, which are, in my opinion, faulty in numerous respects.

First of all, the Grace experts understate the causal relationship between asbestos and mesothelioma by overestimating the background rates of mesothelioma. Both Dr. Moolgavkar and Dr. Weill quote a report by Price and Ware[1] to suggest that the background rate of mesothelioma is 2-4 cases per million population per year. This is based on the rates of mesothelioma in women and assumes that these mesotheliomas are uncommonly (or not at all) asbestos-related. However, earlier studies by McDonald *et al.* concluded that the background rate is 1-2 cases per million population per year.[2] My own studies have shown that at least half of mesotheliomas in women are asbestos-related, and that the threshold is therefore closer to one case per million population per year than to two.[3,4] Similarly, Hillerdal states that if a background rate of mesothelioma exists, it is well below one case per million population per year.[5]

Secondly, Grace experts greatly exaggerate the level of asbestos necessary to cause mesothelioma. Anderson estimates that there is a doubling of mesothelioma risk at a level of 79 fibers/cc-yrs for chrysotile asbestos exposure. However, the correct level is no more than 7-10 fiber/cc/-yrs based on studies by McDonald *et al.* and by Berman and Crump. McDonald *et al.* reported a rate of mesothelioma in Canadian chrysotile miners and millers of 300 cases per million population per year.[6] The average exposure to these workers was between 1000 and 1500 fiber/cc-yrs. Simple arithmetic extrapolation indicates a doubling of the risk of mesothelioma from Canadian chrysotile at a level of 7-10 fiber/cc-yrs, roughly one-tenth the level assumed by Anderson. A risk analysis performed for the EPA by Berman and Crump found even lower levels for chrysotile to produce one case per million person-years.[7] Dr. Anderson is well aware of the Berman and Crump analysis, but inexplicably ignores it.

In addition, both Dr. Anderson and Dr. Moolgavkar understate the mesothelioma risk of asbestos exposure when they assert that there is no convincing evidence for an increased risk of mesothelioma at levels below 15 fiber/cc-yrs. This is astonishing for several reasons. First of all, Dr. Moolgavkar equates the potency of Libby tremolite with that of amosite or crocidolite. This is significant because Hodgson and Darnton, upon whom several other Grace experts rely,[8] find amosite and crocidolite to be 100 and 500 times more potent than chrysotile, respectively. Combining these results with the findings of McDonald *et al.* noted above would indicate a doubling of mesothelioma risk at an amphibole exposure level of no more than 0.1 and perhaps as little as 0.01 fiber/cc/yr. Moreover, multiple studies have found evidence of increased mesothelioma risk below 15 fiber/cc-yrs, in spite of the difficulties involved with doing such low level studies. Investigators of the Wittenoom cohort reported evidence of an increased risk of mesothelioma at cumulative levels of 0.53 fiber/cc-yr.[9] Rodelsperger *et al.* in Europe reported an increased risk at levels of exposure between 0-0.15 fiber/cc-yr.[10] Others have also reported increased risks with low level exposures.[5, 11] Considering the potency of crocidolite and amosite at producing mesothelioma and the fact that no threshold level for mesothelioma has *ever* been identified, I am mystified by Dr. Anderson's and Dr. Moolgavkar's suggestion that there is no evidence of an increased risk of mesothelioma at levels below 15 fiber/cc-yrs. It should be noted that Dr. Moolgavkar's benchmark level for mesothelioma of 3.2 fiber/cc/yrs is almost 100 times Dr. Anderson's benchmark level for background asbestos exposure of 0.035 fiber/cc/yr.

Dr. Moolgavkar again minimizes the risk of asbestos exposure when he opines that a single case of mesothelioma death occurring among Libby, MT residents over the past twenty years could be due to chance alone. There were roughly 2000 residents of Libby who were not employed in the mine or mill. With a background rate of one case per million person-years (see above), contrary to Dr. Moolgavkar's assertion, it would take approximately *500 years* of observation before one case would be expected based on background rates.

Dr. Moolgavkar gives a hypothetical example of a worker who was exposed to 5 fibers/cc of amosite for ten years (50 fiber/cc-yrs) and then develops mesothelioma some 20 years later, and then suggests that depending upon the circumstances of exposure, that

such an exposure may not have been a significant factor. Dr. Moolgavkar is wrong. Based upon the level associated with doubling of mesothelioma risk for Canadian chrysotile from McDonald's work and the 100-fold potency difference between amosite and chrysotile noted by Hodgson and Darnton (see above), the hypothetical exposure is at least 500 times higher than the level associated with doubling of mesothelioma risk as a consequence of amosite exposure. For Dr. Moolgavkar to opine that such an exposure may not be a significant contributing factor to mesothelioma (with the given latency interval) is preposterous. As noted in my prior report, the Peto model *cannot* be properly applied to calculate an attributable fraction of different exposures to an individual patient's mesothelioma.

Dr. Moolgavkar states that 20% to more than 80% of mesotheliomas are not caused by asbestos. Once again Dr. Moolgavkar is wrong. The current consensus is that 80-90% of cases are asbestos-related.[12] Indeed, my own experience in the past ten years based on lung fiber analyses has been that 83% of mesothelioma cases are asbestos-related.[13]

Dr. Moolgavkar suggests that smoking may cause mesothelioma. In fact, there is a scientific consensus that smoking does not cause mesothelioma. [12, 14-17] Although smoking interferes with asbestos fiber clearance from the lower respiratory tract by inhibiting ciliary motion, smoking also decreases the diameter of airways and increases the thickening of the mucous blanket.[18] These two mechanisms are oppositional, so that smoking could either aggravate or diminish the risk of mesothelioma. Only epidemiological studies could resolve the issue, and epidemiology does not indicate any association between mesothelioma and smoking. There is scientific consensus on this issue.

Dr. Moolgavkar states that it is hard to envisage how asbestos could reach the testis. He is apparently referring to mesotheliomas of the tunica vaginalis testis, which is simply an extension of the peritoneum into the scrotum.[12] Asbestos fibers that could reach the peritoneal cavity could have access to this extension of the peritoneum.

Minimum Causation Criteria for Lung Cancer Claims

The medical criteria that Dr. Florence assumes with respect to the minimum causation criteria for lung cancer claims (point 3 at page 2 of the Florence Report) are much too restrictive. Numerous studies show that lung cancer can be caused by asbestos exposure even when the assumed criteria are not satisfied.

Dr. Anderson suggests that there is a doubling of lung cancer risk at 100-278 fiber/cc-yrs. Dr. Moolgavkar also states that the doubling of lung cancer risk occurs at 100+ fiber/cc-yrs. However, the Helsinki criteria concluded that a doubling of lung cancer risk occurs at 25 fiber/cc-yrs, the same threshold that Dr. Anderson identified for asbestosis.[19, 20] Moreover, there is a scientific consensus that populations with asbestosis are at an increased risk of lung cancer.[21]

If Grace's estimates were accurate, then one would expect cohorts with an increased risk of asbestosis for which no increase in lung cancer risk has been documented. I am unaware of any such studies. To the contrary, there have been studies showing an increased risk of lung cancer in asbestos-exposed populations in which asbestosis has not been documented.[22]

Dr. David Weill notes that a critique of the Helsinki criteria has recently been published. The authors of this non-peer-reviewed editorial included Dr. Allen Gibbs, Dr. Richard Attanoos, Dr. Andrew Churg, and Dr. Hans Weill.[23] These authors assert that the Helsinki criteria are slanted toward a particular medicolegal viewpoint. However, it is Drs. Gibbs, Atanoos, Churg, and Weill that have a slanted viewpoint. They fail to state that in asbestos litigation involving lung cancer, they have all testified almost exclusively (if not exclusively) for defendants. They assert that the presence of asbestosis remains the most reasonable criterion for causal attribution purposes. However, they cannot even agree among themselves what criteria for asbestosis should be applied. Dr. Weill requires a clinical-radiographic diagnosis, which excludes cases in which the diagnosis is made pathologically but not clinically (as does the Grace criteria).[24] Drs. Gibbs and Attanoos exclude grade 1 asbestosis as defined by the CAP-NIOSH criteria (modified by Roggli and Pratt), which have been the most widely used criteria for diagnosing asbestosis.[25, 26] And Dr. Churg will diagnose asbestosis based on the finding of a single asbestos body in histological sections showing diffuse lung fibrosis,[27] while Gibbs and Attanoos will not. As noted in my prior report, the extensive studies by Henderson *et al.* as well as the Helsinki consensus document do not require the finding of asbestosis to associate a lung cancer with prior asbestos exposure.[19, 20, 28]

In an attempt to minimize the causal link between asbestos and lung cancer, Dr. Moolgavkar opines that there is disagreement in the scientific community regarding the synergistic effect between cigarette smoking and asbestos exposure in the production of lung cancer. In fact, however, there is scientific consensus on this issue as well. For example, Henderson *et al.* reviewed 16 studies from the medical literature that examined this issue of synergism.[28] Twelve of these studies found that the interaction was multiplicative and two concluded that it was intermediate (between multiplicative and additive). One additional study found intermediate results for women and additive results for men. The final study found that the interaction was additive. Clearly, there is a consensus on this issue: the relationship is multiplicative (*i.e.*, synergism).

Dr. David Weill asserts that the prospective cohort design of the Hughes and Weill[24] study was more powerful than other studies. However, an analysis by Henderson *et al.* in fact showed that the Hughes and Weill study did not have the statistical power to detect a lesser risk of lung cancer in subjects without asbestosis.[28] It is of interest that in a subsequent presentation, Dr. Hans Weill reported that the relative risk of lung cancer in patients with pleural plaques alone (no radiographic evidence of asbestosis) was 1.4. Although that particular study was not statistically significant, a larger and statistically more powerful study by Hillerdal also found a risk of lung cancer of 1.4 among patients with plaques alone, and this finding *was* statistically significant.[29] Thus, Dr. David Weill's conclusion is incorrect.

Minimal medical criteria for Other Cancer claims: Laryngeal Cancer

Dr. David Weill inexplicably concludes that there is insufficient scientific evidence to infer a causal relationship between asbestos exposure and causation of laryngeal cancer. Dr. Weill is wrong as shown by the Asbestos Committee of the Institute of Medicine of the National Academies, which recently addressed the issue of asbestos exposure and causation of laryngeal cancer.[30] That Committee reviewed 35 cohort studies and 15 case-control studies in making their assessment, and concluded that the evidence is *sufficient* to infer a causal relationship between asbestos exposure and laryngeal cancer. Indeed, even Dr. Florence accepts laryngeal carcinoma as an illness amenable to compensation.

Dr. Weill's extreme position raises issues regarding the credibility of Dr. Weill's opinions in general. In his analysis, Dr. Weill failed to consider that three independent pathologic studies found a statistically significant association between parietal pleural plaques and laryngeal cancer.[31-33] Of further note, the lining of the larynx is similar to the lining of the bronchial tree, for which there is no doubt that asbestos is carcinogenic. Studies have shown that the surface area of the bronchial tree is roughly 2500 cm^2,[34] whereas that of the larynx is no more than 25 cm^2. If occurrence of cancer in the respiratory tract is random regarding which epithelial cell will become transformed to malignancy, one would expect 100 lung cancers for each laryngeal cancer that developed. Selikoff's data show that this ratio is far less than 100:1.[35]

Minimum criteria for asbestosis: $FEV_1/FVC \geq 65\%$

Grace's requirement of FEV1/FVC ≥65% for a causal link between asbestos exposure and asbestosis is wrong.

One of the cases studied in my laboratory was a 57-year-old man who died of respiratory failure. At autopsy, he had severe emphysema and grade 3 asbestosis on a scale of 0-4. An analysis of lung tissue demonstrated hundreds of thousands of times the expected or background amount of asbestos. He was an insulator for 30 years. And yet this man would not receive compensation under the Grace criteria because of the obstructive disease from emphysema superimposed upon his restrictive disease from asbestosis (*i.e.*, $FEV_1/FVC < 65\%$). There are many additional cases such as this, and they underscore the over-restrictiveness and unfairness of the Grace minimum exposure criteria.

It is well recognized that asbestos exposure causes peribronchiolar fibrosis, which some investigators have termed mineral dust airways disease since other mineral dust besides asbestos can also cause this lesion. Dr. Weill states that the clinical importance of peribronchiolar fibrosis is unknown. Churg and Wright, who actually studied this lesion, concluded that these pathologic changes are most likely associated with chronic airflow obstruction.[36] Furthermore, Churg *et al.* concluded that mineral dust airways

disease is associated with abnormalities of airflow greater than those induced by smoking alone.[37]

Additional Ways in Which Grace's Experts Underestimate the Risk of Asbestos Exposure

Grace's experts underestimate the risk of asbestos exposure in many other respects as well.

Dr. Weill states that there is no causal relationship between parietal pleural plaques and mesothelioma. However, my studies have shown that more than half of patients with mesothelioma have pleural plaques.[13] Similarly, Dr. Hillerdal has reported an eleven-fold increased risk of mesothelioma in patients who have plaques.[29] The Helsinki criteria recognize plaques as a marker for asbestos causation in mesothelioma cases.[19] My own studies have shown that in 65 consecutive patients with pleural plaques and mesothelioma, there was an elevated tissue asbestos content (unpublished observations).

Dr. Moolgavkar also states that phase contrast light microscopy (PCM) can only detect fibers > 0.4 μm in diameter. However, PCM can detect fibers down to 0.2 μm in diameter.[38]

Dr. Moolgavkar claims that crocidolite is cleared much more slowly than amosite from the respiratory tract. I do not believe that this claim is supported by animal studies.[39] Furthermore, Churg estimates that the half-life of commercial amphibole fibers in the human lung is 10-20 years, and does not make a distinction according to fiber type.[27, 40]

Finally, Dr. Steven E. Haber, in his review of the Darlene J. Riley case, notes that the pathology was reviewed by Dr. Colby, and then recommends that an expert in lung pathology should review the pathology material in this case. Dr. Colby is not only one of the foremost lung pathologists in the United States and the world, but is also a member of the U.S. Canadian and the International Mesothelioma Panels.

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REFERENCES:

1. Price B, Ware A. Mesothelioma trends in the United States: an update based on Surveillance, Epidemiology and End Results Program data from 1973 through 2003. Am J Epidemiol 2004; 159: 107-12.

2. McDonald AD, McDonald JC. Malignant mesothelioma in North America. Cancer 1980; 46: 1650-6.

3. Roggli VL, Oury TD, Moffatt EJ. Malignant mesothelioma in women. In: Anatomic Pathology, 1997, Vol. 2 (Rosen PP, Fechner RE, eds.), ASCP Press, Chicago, 1998, pp. 147-63.

4. Roggli VL, Sharma A, Butnor KJ, Sporn T, Vollmer RT. Malignant mesothelioma and occupational exposure to asbestos: A clinicopathological correlation of 1445 cases. Ultrastruct Pathol 2002; 26: 55-65.

5. Hillerdal G. Mesothelioma: cases associated with non-occupational and low dose exposures. Occup Environ Med 1999; 56: 505-13.

6. McDonald AD, Case BW, Churg A, Dufresne A, Gibbs GW, Sebastien P, McDonald JC. Mesothelioma in Quebec chrysotile miners and millers: Epidemiology and aetiology. Ann Occup Hyg 1997; 41: 707-19.

7. Berman DW, Crump KS. Technical Support Document for a Protocol to Assess Asbestos-Related Risk: Final Draft. US Environmental Protection Agency #9345.4-06, October 2003.

8. Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. Ann Occup Hyg 2000; 44: 565-601.

9. Hansen J, de Klerk NH, Musk AW, et al. Environmental exposure to crocidolite and mesothelioma: exposure-response relationships. Am J Respir Crit Care Med 1998; 157: 69-75.

10. Rodelsperger K, Jockel K-H, Pohlabeln H, Romer W, Woitowitz H-J. Asbestos and man-made vitreous fibers as risk factors for diffuse malignant mesothelioma: Results from a German hospital-based case-control study. Am J Ind Med 2001; 38: 1-13.

11. Iwatsubo Y, Pairon JC, Boutin C, Menard O, Massin N, Callaud D, Orlowski E, Galateau-Salle F, Bignon J, Brochard P. Pleural mesothelioma: Dose-response relation at low levels of asbestos exposure in a French population-based case-control study. Am J Epidemiol 1998; 148: 133-42.

12. Sporn TA, Roggli VL. Mesothelioma, CH 5, In: *Pathology of Asbestos-Associated Diseases, 2nd Ed.* (Roggli VL, Oury TD, Sporn TA, eds.), Springer: New York, 2004, pg. 104.

13. Roggli VL, Vollmer RT. Twenty-five years of fiber analysis: What have we learned? Hum Pathol (in press, 2007).

14. Browne K. Asbestos-related disorders. CH 14, In: Occupational Lung Disorders, 3rd Ed. (Parkes WR, ed.), Butterworth-Heineman: Oxford, 1994, pp. 411-504.

15. Hammar SP. Pleural diseases, CH 34, In: Pulmonary Pathology, 2nd Ed (Dail DH, Hammar SP, eds.), Springer-Verlag: New York, 1994, pp. 1463-579.

16. Churg A. Neoplastic asbestos-induced disease. CH 10, In: Pathology of Occupational Lung Disease, 2nd Ed. (Churg A, Green FHY, eds.), Williams & Wilkins: Baltimore, 1998, pp. 339-91.

17. de Klerk NH, Musk AW. Epidemiology of mesothelioma. CH 19, In: Mesothelioma (Robinson BWS, Chahinian AP, eds.), Martin Dunitz: London, 2002, pp. 339-49.

18. Hessel PA, Sluis-Cremer GK, Lee SL. Distribution of silicotic collagenization in relation to smoking habits. Am Rev Respir Dis 1991; 144: 297-301.

19. Henderson DW, Rantanen J, Barnhart S, Dement JM, De Vuyst P, Hillerdal G, Huuskonen MS, Kivisaari L, Kusaka Y, Lahdensuo A, Langard S, Mowe G, Okubo T, Parker JE, Roggli VL, Rödelsperger K, Rösler J, Tossavainen A, Woitowitz HJ. Asbestos, asbestosis, and cancer: The Helsinki criteria for diagnosis and attribution. A consensus report of an international expert group. *Scand. J. Work Environ. Health* **23**:311, 1997.

20. Henderson DW, Rodelsperger K, Woitowitz H-J, Leigh J. After Helsinki: a multidisciplinary review of the relationship between asbestos exposure and lung cancer, with emphasis on studies published during 1997-2004. Pathology 2004; 36: 517-50.

21. Roggli VL. Carcinoma of the Lung, CH 7, In: *Pathology of Asbestos-Associated Diseases, 2nd Ed.* (Roggli VL, Oury TD, Sporn TA, eds.), Springer: New York, 2004, pg. 193.

22. Wilkinson P, Hansell DM, Janssens J, Rubens M, Rudd RM, Taylor AN. Is lung cancer associated with asbestos exposure without small opacities on the chest radiograph? Lancet 1995; 345: 1074-8.

23. Gibbs A, Attanoos RL, Churg A, Weill H. The "Helsinki criteria" for attribution of lung cancer to asbestos exposure: How robust are the criteria? Arch Pathol Lab Med 2007; 131: 181-4.

24. Hughes JM, Weill H. Asbestosis as a precursor of asbestos related cancer: results of a prospective mortality study. Br J Ind Med 1991; 48: 229-33.

25. Craighead JE, Abraham JL, Churg A, et al. The pathology of asbestos-associated diseases of the lungs and pleural cavities: Diagnostic criteria and proposed grading schema. (Report of the Pneumoconiosis Committee of the College of American Pathologists and the National Institute for Occupational Safety and Health) Arch Pathol and Lab Med 1982; 106: 544-96.

26. Roggli VL, Pratt PC. Asbestosis, CH 4, In: Pathology of Asbestos-Associated Diseases (Roggli VL, Greenberg SD, Pratt PC, eds.), Little, Brown & Co: Boston, 1992, pp. 77-108.

27. Churg A. Nonneoplastic disease caused by asbestos, CH 9, In: Pathology of Occupational Lung Disease, 2nd Ed. (Churg A, Green FHY, eds.), Williams & Wilkins: Baltimore, 1998, pp. 277-338.

28. Henderson DW, de Klerk NH, Hammar SP, Hillerdal G, Huuskonen MS, Karjalainen A, Leigh J, Pott F, Roggli VL, Shilkin KB, Tossavainen A. Asbestos and lung cancer: Is it attributable to asbestosis or to asbestos fibre burden? Chapter 6, In: *Pathology of Lung Tumors* (Corrin, B., ed.), Churchill Livingstone, London, 1997, pg. 83.

29. Hillerdal G. Pleural plaques and risk for bronchial carcinoma and mesothelioma: A prospective study. Chest 1994; 105: 144-50.

30. Institute of Medicine of the National Academies. Laryngeal cancer and asbestos, CH 8, In: Asbestos: Selected Cancers. The National Academies Press: Washington, DC, 2006, pp. 173-92.

31. Wain SL, Roggli VL, Foster WL. Parietal pleural plaques, asbestos bodies, and neoplasia: A clinical, pathological, and radiographic correlation of 25 consecutive cases. Chest 1984; 86:707-13.

32. Hillerdal G. Pleural plaques and risks for cancer in the county of Uppsala. Eur J Respir Dis 1980; 61 [Suppl 107]: 111-7.

33. Mollo F, Andrion A, Colombo A, Segnan N, Pira E. Pleural plaques and risk of cancer in Turin, Northwestern Italy: An autopsy study. Cancer 1984; 54: 1418-22.

34. Mercer RR, Russell ML, Roggli VL, Crapo JD. Cell number and distribution in human and rat airways. Am Rev Respir Cell Mol Biol 1994; 10: 613-24.

35. Selikoff IJ, Seidman H. Asbestos-associated deaths among insulation workers in the United States and Canada, 1967-1987. Ann NY Acad Sci 1991; 643: 1-14.

36. Churg A, Wright JL. Small airways disease and mineral dust exposure. Pathol Annu 1983; 18: 233-51.

37. Churg A, Wright JL, Wiggs B, Pare PD, Lazar N. Small airways disease and mineral dust exposure: Prevalence, structure, and function. Am Rev Respir Dis 1985; 131: 139-43.

38. Roggli VL, Coin P. Mineralogy of asbestos, CH 1, In: Pathology of Asbestos-Associated Diseases, 2nd Ed. (Roggli VL, Oury TD, Sporn TA, eds.), Springer; New York, 2004, pp. 1-16.

39. Fattman CL, Chu CT, Oury TD. Experimental models of asbestos-related diseases. CH 10, In: Pathology of Asbestos-Associated Diseases, 2nd Ed. (Roggli VL, Oury TD, Sporn TA, eds.), Springer; New York, 2004, pp. 256-308.

40. Churg A, Vedal S. Fiber burden and patterns of asbestos-related disease in workers with heavy mixed amosite and chrysotile exposure. Am J Respir Crit Care Med 1994; 150: 663-9.